**Pediatrics – Dr. Haidar – Lecture 10 - Neonatal Emergencies**

**Shock**

Shock in the delivery room is manifested by cyanosis, pallor, poor capillary refilling time, unpalpable pulses, hypotonia and eventually cardiopulmonary arrest.

Cyanosis in the presence of pallor, with or without purpuric ecchomosis is very suggestive of shock.

**Causes:**

Blood loss prior to or during labor is a common cause of shock in the delivery room, this loss may be due to a fetal-maternal hemorrhage, placenta previa, twin–twin transfusion syndrome or displacement of blood from the fetus to the placenta as during asphyxia. Other causes like bleeding in the viscus such as liver and spleen, hemorrhage into the cerebral ventricles, especially in the preterm infant, also other etiologies such as anemia, hypoalbuminemia, hypovolemia, Rh immune hydrops, and severe intrauterine bacterial sepsis (manifested as mottled skin, hypotonia, cyanosis, diminished peripheral pulses, neutropenia, thrombocytopenia, and disseminated intravascular coagulation).

**Management:**

Approaches should be done according to the expected causes. Hypovolemic shock should be managed with repeated boluses of 10-15 ml/kg of either normal saline or albumin, if hemolysis is suspected so blood should be given (all blood should be cross-matched with both blood of the neonate and mother before administration). In addition to that vasoactive drugs like Dopamin, epinephrine may improve the cardiac output and tissue perfusion.

**Asphyxia**

Fetal or neonatal hypoxia, hypercapnia, poor cardiac output and a metabolic acidosis can result from one or a number of many conditions affecting the fetus, placenta or the mother. Whether in utero or after birth, asphyxia—caused hypoxic ischemic brain injury result from reduced gaseous exchange through the placenta or through the lungs respectively. Asphyxia associated with severe bradycardia or cardiac insufficiency will reduce or eliminate tissue blood flow, resulting in ischemia. The fetal and neonatal circulatory system respond to reduced oxygen availability by shunting the blood preferentially to the brain, heart and the adrenal glands away from intestine, kidney, lung and skin.

The response to hypoxia also is characterized by:

1. Release catecholamines from the adrenal glands.
2. Transient hypertension and tachycardia followed by bradycardia and shock.
3. Production of mixture of metabolic and respiratory acidosis.
4. Hypoxemia.

**Etiology of asphyxia:**

1. Intrauterine causes: like uteroplacental insufficiency, abruptio placenta, prolapsed cord, maternal hypotension, placenta previa, fetomaternal hemorrhage.
2. Intrapartum causes: like cephalopelvic disproportion, shoulder dystocia, breech presentation, umbilical cord compression.
3) Postpartum causes: like maternal medications, trauma, congenital myasthenia gravis, shock, choanal atresia, severe immaturity, pneumothorax, diaphragmatic hernia, lung hypoplasia.

**Effect of Asphyxia:**

1) C.N.S.: hypoxic–ischemic encephalopathy. IVH, cerebral edema, seizures, hypotonia, hypertonia.
2) C.V.S.: myocardial ischemia, poor contractility, hypotension.
3) R.S.: respiratory distress syndrome, persistent fetal circulation.
4) Renal system: acute tubular or cortical necrosis.
5) Adrenal: adrenal hemorrhage.
7) Metabolic: hyponatremia, hypocalcemia, hypoglycemia, syndrome of inappropriate ADH.
8) Hematology: D.I.C.

**Hypoxic–ischemic encephalopathy:**

Typically H.I.E. in the term infant is characterized by cerebral edema, cortical necrosis, and involvement of the basal ganglia, where in the preterm infant it is characterized by periventricular leukomalacia. Both lesions may result in cortical atrophy, mental retardation and spastic quadriplegia or diplegia.

**Clinical Features:**

Vary according to the severity of the injury, infants with severe diseases are usually hypotonic, although occasionally they appear hypertonic and hyperalert at birth. As cerebral edema develops (due to brain anoxia), brain functions become affected producing coma, apnea, and as edema progresses produce refractory seizures (begins between 12-24 hr after birth) there will be also hypotonia, diminished or absence of deep tendon reflexes.

**Criteria of Asphyxia as a cause of brain injury:**

- Profound metabolic or mixed acidemia (PH < 7.00) on umbilical cord arterial blood sample.
- Persistent of an Apgar score of 0-3 for longer than 5 minutes.
- Neonatal neurological sequelae (e.g., seizures, coma, or hypotonia).
- Multiorgan system dysfunction (e.g., renal, C.V.S., G.I.T., R.S., or hematologic disorders).

**Treatment:**

1) General therapy: restore oxygen supply to the body tissues, especially the brain. This requires ventilation with oxygen and ensuring an adequate cardiac output. The secondary objective is to evaluate the degree of hypoxic injury and to plan treatment.

2) Specific therapy: according to the severity of the disease and this include drugs that decreasing the brain edema like steroid, manitol in addition to the routine therapy in N.I.C.U. and also including treatment of any complications that may occur.
**Prognosis:**

Outcome is related to the severity and duration of the asphyxial insult and to the adequacy of compensatory mechanisms, resuscitation procedures and specific treatment of multiorgan system involvement. Neurological outcome is the most difficult to predict but is the best related to the degree of the hypoxic encephalopathy and EEG activity in the neonatal period, and to findings on physical examination of the infant at 9-12 months of age.

**Apnea**

Apnea: is defined as a cessation of breathing for longer than 15-20 sec, or a respiratory pause of any duration if accompanied by bradycardia and cyanosis or oxygen desaturation as evidence by pulse oximetry monitoring.

**Categories of apnea:**

1) Central apnea: describes a complete cessation of chest wall movements and no airflow.
2) Obstructive apnea (apnea secondary to airway obstruction): describes chest wall movements or respiratory efforts but without airflow. Commonly available apnea monitors do not record obstructive apnea because they continue to detect chest wall movements.
3) Mixed apnea: is a combination of central and obstructive apnea and constitute the most frequent type encountered in preterm infants.

**Causes of neonatal apnea:**

1) C.N.S. causes: IVH, drugs, seizures, hypoxic injury.
2) Respiratory causes: pneumonia, obstructive airways lesions, atelectasis, extreme prematurity, laryngeal reflex, RDS.
3) Infections: sepsis, necrotizing enterocolitis, meningitis.
4) G.I.T: oral feeding, bowel movement, GER, intestinal perforation.
5) Metabolic: hypoglycemia, hypocalcemia, hypothermia.
6) C.V.S: hypotension, hypertension, heart failure, anemia, hypovolemia.
7) Idiopathic.

**Idiopathic apnea of prematurity:** apnea that occur in preterm infants and reflects immaturity of the respiratory control mechanisms in the brain stem. It is frequently increases with decreasing gestational age. Incidence is as high as 85% in infants <28 wk gestation and 25% in infants 33-34 wk gestation.

**Clinical features:** it occur in the absence of any identifiable cause, usually appearing 24 hr after birth and during the first week of life. It usually resolves by postconceptional age of 38-44 wk, it is usually characterized by apnea that associated with bradycardia (HR < 80 beats/min). Apnea of prematurity is characterized by periodic breathing and intermittent hypoxia, which further diminish respiratory drive.

**Management:**

idiopathic apnea of prematurity is a diagnosis of exclusion, and therefore a search for underlying causes must be undertaken.
Management principle include:

1- maintenance of a neutral thermal environment, treatment of hypoxia, and proprioceptive stimulation.

2- respiratory stimulant medication as needed (caffeine or theophylline).

3- ventilation as needed (by bag and mask).

4- CPAP or mechanical stimulation.